Aortic root enlargement: What are the operative risks?

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Objective: Despite concern that small aortic valve prostheses can lead to prosthesis–patient mismatch with diminished left ventricular mass regression and poor long-term outcome after aortic valve replacement, there remains reluctance to perform aortic root enlargement procedures. We therefore examined the operative risks of aortic valve replacement with and without root enlargement.

Methods: We reviewed perioperative outcomes among patients undergoing aortic valve replacement at our institution between January 1993 and December 2001. Risk factors for operative death were evaluated by means of multivariable analysis.

Results: Of 2366 patients undergoing aortic valve replacement with (1173) or without (1193) concomitant procedures, 249 (10.5%) underwent posterior root enlargement. Patients undergoing complex root enlargement (Konno–Rastan procedures) were excluded. Patients undergoing aortic root enlargement were significantly younger, twice as often female, and more often undergoing a reoperation but were similar with respect to functional class. The mean valve implant size was less in the aortic root enlargement group (21.5 ± 1.6 vs 23.2 ± 2.3 mm, $P < .0001$). As expected, mean crossclamp time and bypass time were somewhat longer with root enlargement. Raw operative mortality was higher with aortic root enlargement (5.6% vs 2.9%, $P = .0324$); however, by means of multivariable analysis, advanced functional class ($P = .0020$; odds ratio, 1.87), preoperative congestive heart failure ($P < .0001$; odds ratio, 3.22), and smaller valve implant size ($P = .012$; odds ratio, 1.16), but not aortic root enlargement, were independent risk factors for operative death.

Conclusions: Aortic root enlargement itself does not increase operative risk, although it is most often required among high-risk patients. Surgeons should not be reluctant to enlarge the aortic root to permit implantation of adequately sized valve prostheses.

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The surgical management of the small aortic root accordingly remains a relevant topic. It is intuitive that one would elect to replace a stenotic valve (or for that matter a regurgitant valve) with the least stenotic prosthesis. Therefore, it is not surprising that a number of studies have demonstrated superior left ventricular mass regression,[1] postoperative functional class and exercise tolerance,[2] and patient survival[3] when small valves are avoided. Furthermore, prosthesis–patient mismatch (PPM)[4] specifically has been shown by some investigators to adversely affect left ventricular mass regression[5,6] and both early and late survival.[7-10]

Other authors dispute the relevance of PPM in the current era, reporting little or no relationship between valve orifice size and outcome.[11,12] It has been further suggested that PPM is, in practice, quite uncommon.[13] These arguments

Abbreviations and Acronyms

ARE = aortic root enlargement
AVR = aortic valve replacement
iEOA = indexed effective orifice area
PPM = prosthesis–patient mismatch
STS = Society of Thoracic Surgeons

are complicated by various definitions of PPM ranging from an indexed orifice area of less than 0.6 cm^2/m^2,[13] to less than 0.85 cm^2/m^2,[11,12] as well as dispute over the more appropriate measure of orifice area (geometric or effective).

Although some physicians continue to debate the clinical effect of aortic valve prosthesis size on outcome, interest in prosthetic hemodynamics persists. Indeed, superior hemodynamic performance is the very basis of many arguments in favor of the use of stentless xenografts and the Ross pulmonary autograft operation. Furthermore, hemodynamic improvements remain a common selling point among valve manufacturers, with each new-generation valve promising superior flow characteristics.

Regardless of academic argument, the practicing surgeon has a number of options available when confronted with the small aortic root and a circumstance in which he or she wishes to implant a valve larger than the annulus readily accepts. Among those options is posterior aortic root enlargement (ARE). Many surgeons are reluctant to perform ARE, however, out of concern that this adjunctive procedure will increase operative morbidity and mortality.[14] This approach, however, has been our institutional preference for management of the small annulus since Stenseth and colleagues[15] first introduced it as an approach to prevent tertiary orifice obstruction after implantation of the Starr–Edwards prosthesis. We find unappealing the more complex alternatives promulgated today using stentless xenografts, homografts, or autografts as full root replacements, a procedure associated with an almost 3-fold higher operative risk than simple aortic valve replacement (AVR) in the Society of Thoracic Surgeons (STS) database (http://www.sts.org).[16] We therefore reviewed our experience with ARE among patients undergoing AVR with or without concomitant procedures.

Materials and Methods

Patient Population

After review and approval by the Mayo Clinic Rochester Institutional Review Board, we retrospectively reviewed the clinical records of 2366 consecutive adult patients undergoing AVR from January 1993 through December 2001. Of these, 249 (10.5%) underwent posterior root enlargement. Only those cases in which the operative note documented use of patch material to accomplish root enlargement, as opposed to its use to facilitate closure of a calcified or otherwise complicated aortotomy, were included. Patients undergoing Konno–Rastan procedures were excluded, as were patients undergoing concomitant mitral valve repair or replacement, aortic aneurysm repair, or composite aortic root reconstruction. Those undergoing concomitant coronary artery bypass grafting were included because they not only constituted a large fraction of both groups but also reflect the patient population in which the clinical decision to enlarge the annulus must be made in practice. Patients with other miscellaneous concomitant procedures, such as tricuspid valve repair and atrial septal defect closure, the effect of which on mortality was deemed negligible, were included. All clinical data were collected prospectively according to the guidelines and definitions of the STS database.

Surgical Technique

Operations were performed routinely under normothermic cardiopulmonary bypass with intermittent cold blood cardioplegia. There is no uniform policy in our unit with regard to minimum acceptable prosthesis size, and opinions vary among surgeons. If, however, the aortic annulus will not accept the valve size the surgeon believes to be appropriate for the given patient, it is our general preference to enlarge the annulus rather than implant a stentless xenograft valve or homograft.

There is considerable confusion in the literature regarding the techniques proposed by the eponymous descriptors Nicks'[7] and Manouguian's[17] enlargement. Key figures from the original contributions are therefore reproduced in Figure 1. Our technique is similar to that outlined by Nicks and associates.[7] Our standard aortotomy for AVR is oblique, extending into the noncoronary sinus. If the aortic annulus will not accommodate the desired prosthesis, this aortotomy can be extended to but not beyond the annulus because widening the apex with pericardium permits implantation of a slightly larger valve by slightly tilting the prosthesis such that the prosthetic sewing ring rides above the native annulus and is secured to the patch itself.

A somewhat greater enlargement can be accomplished by extending the incision in the base of the noncoronary sinus beyond the aortic annulus onto the anterior mitral leaflet (Figure 2, *A*). Once across the annulus, the incision is directed posteriorly toward the center of the anterior leaflet of the mitral valve. A teardrop-shaped patch of autologous or bovine pericardium is sutured to the base of the incision with a 4-0 polypropylene suture, with the broader end of the patch at the apex. After completion of the suture line above the level of the divided annulus, the valve prosthesis is sutured in place in a supra-annular position with 2-0 pledgeted Ethibond (Ethicon, Somerville, NJ) mattress sutures. In the region of the patch, the sutures are passed full thickness from outside the patch to inside. The pericardial patch is then used to facilitate closure of the aortotomy (Figure 2, *B*).

As a cautionary technical note, there is potential for impingement of leaflet motion when a bileaflet mechanical prosthesis is implanted with this technique. Leaflet mobility must be conscientiously inspected. Rotation of the prosthesis to obtain optimal valve closure is not uncommon.

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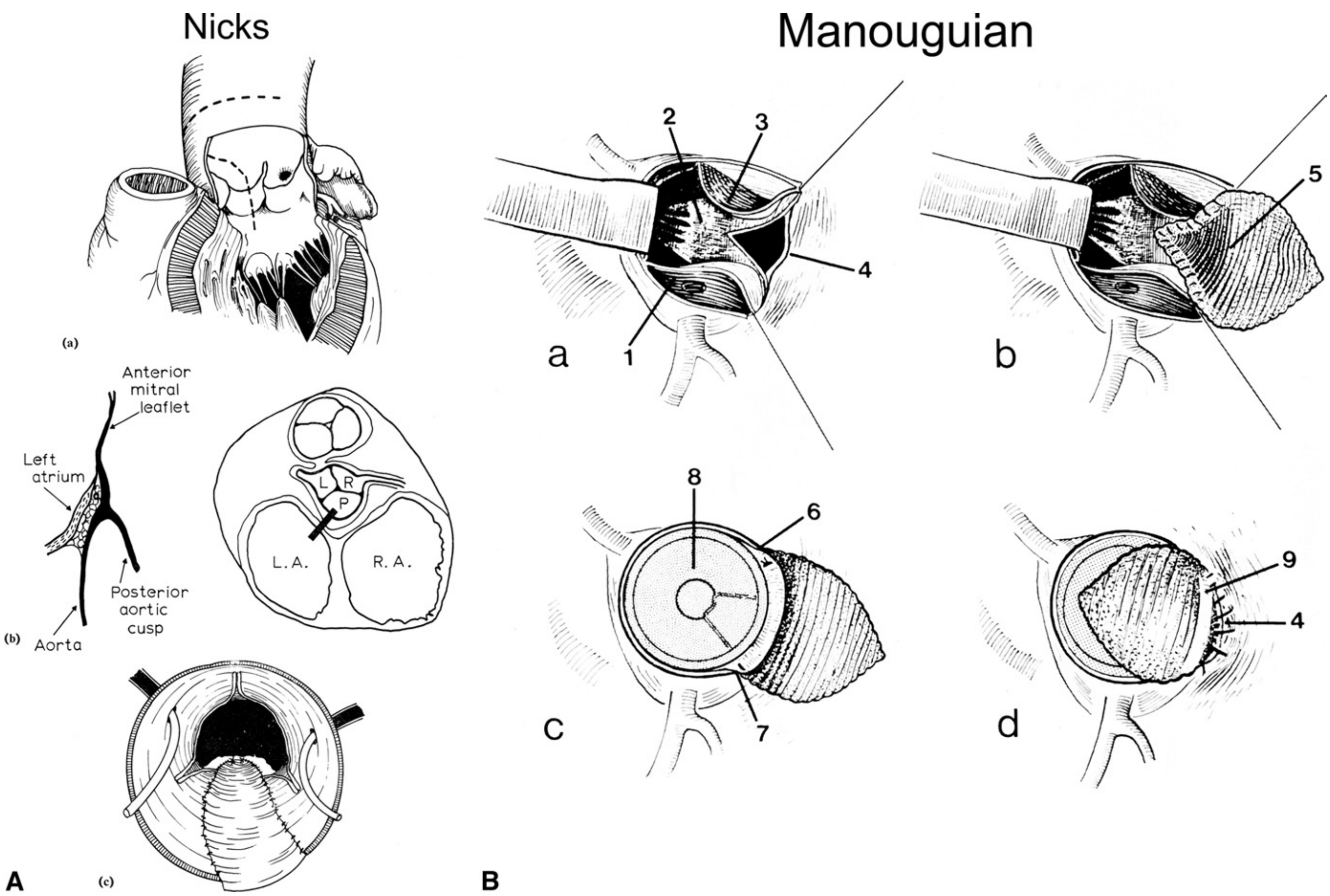

Figure 1. Original figures from articles published by Nicks and colleagues (A) and Manouguian and Seybold-Epting (B) first describing posterior root enlargement techniques. Figure 1A is reprinted from Nicks R, Cartmill T, Bernstein L. Hypoplasia of the aortic root. The problem of aortic valve replacement. *Thorax*. 1970;25:339-46. Reproduced with permission from the BMJ Publishing Group. Figure 1B is reprinted from Manouguian S, Seybold-Epting W. Patch enlargement of the aortic valve ring by extending the aortic incision into the anterior mitral leaflet. New operative technique. *J Thorac Cardiovasc Surg*. 1979;78:402-12. Reproduced with permission from Mosby.

Statistical Analysis

Categoric factors were compared between groups by using the Fisher exact test, whereas continuous factors were compared by using Wilcoxon rank–sum tests. Univariate and multivariate risk factors for operative mortality were evaluated by using logistic regression analysis. The final multivariate model was constructed with a stepwise selection technique.

Results

The clinical characteristics of the 2117 patients undergoing AVR without ARE (AVR group) and the 249 patients undergoing AVR with ARE (ARE group) are shown in Table 1. The mean age in the ARE group was somewhat lower than that of control subjects, and fewer had an ejection fraction of less than 35%. The latter might in part be due to the higher incidence of aortic regurgitation in the AVR group. Patients undergoing ARE were twice as often female, however, and they had more often undergone prior cardiac operations than had the control subjects. Both groups were similar with regard to their functional class and the presence of endocarditis.

As might be anticipated, both aortic crossclamp time and cardiopulmonary bypass time were modestly prolonged in the ARE group (Table 2). Although other concomitant procedures were more common in the AVR group, ARE appeared to add only approximately 10 minutes to the mean myocardial ischemic time. Mechanical valves were more often implanted in the ARE group, perhaps reflecting the somewhat younger age of those

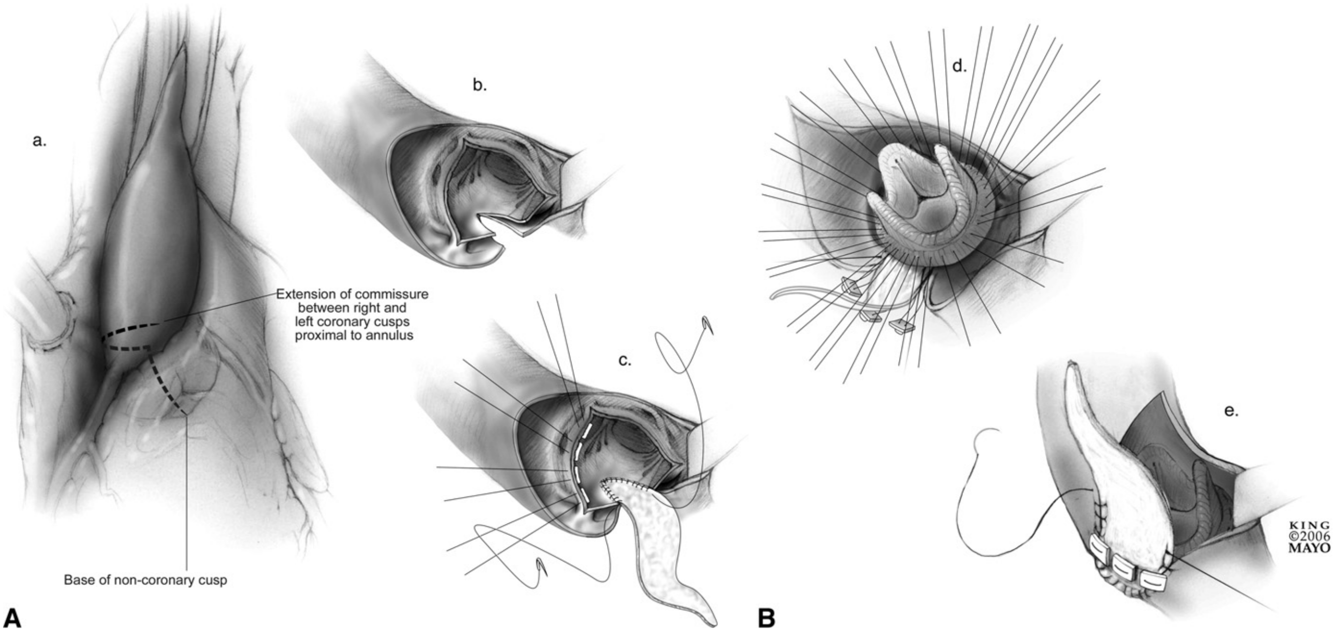

Figure 2. A: a, Nicks' aortic root enlargement is accomplished by extending the aortotomy incision across the aortic annulus into the anterior leaflet of the mitral valve. b, Care must be taken to carry this incision posteriorly into the center of the anterior leaflet. c, The defect is closed with autologous or bovine pericardium. Figure 2B: d, Valve prosthesis is sutured in a supra-annular position. In the region of the patch, the sutures are passed full thickness from outside the patch to inside. e, The pericardial patch is then used to facilitate closure of the aortotomy.

patients and the superior hemodynamic results obtained with these valves.

As shown in Table 3, the incidence of postoperative stroke was similar in both groups, as was the incidence of reoperation for bleeding. Raw operative mortality, however, was higher in the ARE group (5.6% vs 2.9%, $P = .0324$). When only isolated AVR procedures were considered, the difference was slightly less and no longer statistically significant (4.5% vs 2.2%, $P = .0953$), although the trend remained.

Variables enlisted in the univariate and multivariate model for operative mortality are shown in Table 4. Crossclamp time and bypass time were not included because it was expected that these would be strongly associated with ARE. Univariate predictors of operative mortality included ARE procedure, as well as age, sex, body surface area, and valve implant size, in addition to New York Heart Association functional class and history of congestive heart failure. By means of multivariate analysis, however, only smaller valve implant size, preoperative functional class, and congestive heart failure remained significant. When both groups were matched for these significant variables, ARE was not a risk factor for death.

Because the clinical decision regarding root enlargement is made among patients with small roots and prosthetic size itself was shown to be a risk factor in our study, we made an effort to match cohorts with comparative valve sizes. When a subgroup of patients with small valves were considered (Table 5, *A*), the difference in mortality rate was not statistically significant (7.1% vs 4.5%, $P = .2139$), although again the trend remained. Because performing an ARE permits implantation of a 1-size-larger valve, we also compared the group of patients with ARE and a valve size of less than 23 mm with the subset of patients in the AVR group having 1 valve size smaller (ie, <21 mm). The trend for a difference in mortality was smaller (6.0% vs 4.5%) and again not statistically significant (Table 5, *B*).

Discussion

Our data demonstrate that posterior ARE can be accomplished during AVR without significantly increasing operative risk. Additionally, our findings confirm small valve size as an independent risk factor for operative mortality. Taken together, these findings support the continued value of this approach as an option when a surgeon

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Table 1. Preoperative characteristics

Variable	ARE	AVR	*P* value
No.	249	2117	
Mean age (y)	64.3 ± 19.6	70.1 ± 12.2	.006
Sex			
Male	72 (28.9%)	1408 (66.5%)	
Female	177 (71.1%)	709 (33.5%)	<.0001
BSA (m^2)	1.92 ± 1.20	1.98 ± 0.25	<.0001
Prior cardiac operations (redo)	78 (31.3%)	118 (5.6%)	<.0001
NYHA class			
1	9 (3.6%)	129 (6.1%)	
2	39 (15.8%)	433 (20.5%)	
3	171 (69.2%)	1250 (59.3%)	
4	28 (11.4%)	297 (14.1)	.1964
Endocarditis	2 (0.9%)	48 (2.3%)	.1622
LVEF <35%	14 (5.6%)	217 (10.6%)	.0136
Aortic valve lesion			
Stenosis	84 (33.7%)	388 (18.3%)	<.0001
Insufficiency	10 (4.0%)	233 (11.0%)	.0002
Mixed	153 (61.5%)	1482 (70%)	.0072

ARE, Aortic root enlargement; *AVR*, aortic valve replacement; *BSA*, body surface area; *NYHA*, New York Heart Association; *LVEF*, left ventricular ejection fraction.

wishes to implant a larger prosthesis than the native aortic annulus would otherwise accept.

These findings are consonant with those of other authors. Sommers and David[14] observed a statistically insignificant trend toward a higher mortality rate among patients undergoing ARE (7.1% vs 3.5%); however, subsequent studies by both Castro and colleagues[18] and Kitamura and associates[19] reported mortality rates among patients undergoing ARE that were actually lower than those observed among patients undergoing isolated AVR (2.5% vs 4.3% and 3.6% vs 5.9%, respectively). In none of these studies did multivariate analysis identify ARE as a risk factor for operative death.

Our findings also confirm the observations of others that small valve size is itself a risk factor for operative death,[8,20,21] which might in part explain a higher observed mortality rate in the ARE group compared with the AVR group because the procedure is required among patients with a small annulus. The concept of PPM was formally introduced into the literature by Rahimtoola[4] in 1978. In theory, PPM exists to some degree whenever the effective orifice area of the prosthetic valve is less than that of the normal valve. In practice, this is the case, to a greater or lesser extent, with almost all prosthetic options. Indeed, this was the rationale put forward by Nicks and colleagues[7] in their original article on the subject of ARE. The disagreement on the subject concerns the clinical effect of PPM. Some authors argue that PPM rarely occurs,[13] and others argue that even if it is present, it is of no clinical significance.[11,12] Members of the opposing camp cite data indicating inferior left ventricular mass regression[5,6] and reduced long-term survival[9,10,20] among patients with PPM. Our data do not, however, address directly the issue of PPM, and accordingly, we cannot make statements based on our study results concerning the appropriate application of this technique. We can only document its low risk, particularly compared with that reported to the STS database for full root replacement.[16]

It should also be noted that arguments concerning PPM are complicated by disagreement over its definition. Perhaps not surprisingly, those who define PPM most stringently as an indexed effective orifice area (iEOA) of less than 0.60 cm^2/m^2,[13] report it to be a rare occurrence, whereas others who define it as an iEOA of less than 0.85 cm^2/m^2,[11,12] report little effect on late survival. Even more confusion is engendered by the various uses of effective orifice area and geometric orifice area for

Table 2. Operative details

Variable	All ARE	All AVR	*P* value	Isolated ARE	Isolated AVR	*P* value
Aortic clamp time (min)	75.1 ± 29.8	64.0 ± 26.1	<.0001	70.6 ± 27.5	51.4 ± 19.8	<.0001
CPB time (min)	116.5 ± 53.5	93.6 ± 40.5	<.0001	106.3 ± 47.5	73.9 ± 30.2	<.0001
Concomitant procedures	101 (40.6%)	1072 (50.6%)	.0031			
CABG	85 (34.1%)	1037 (49.0%)	<.0001			
Other	17 (6.8%)	68 (3.2%)	.0102			
Valve implant type						
Mechanical	105 (42.2%)	806 (38.1%)		68 (43.6%)	488 (46.3%)	
Bioprosthesis	144 (57.8%)	1311 (61.9%)	.2157	88 (56.4%)	566 (53.7%)	.5477
Mean ± SD size (range [median])	21.5 ± 1.6 (19-25 [21])	23.2 ± 2.3 (17-33 [23])	<.001	21.6 ± 1.7 (19-25 [21])	23.4 ± 2.4 (17-33 [23])	<.0001

ARE, Aortic root enlargement; *AVR*, aortic valve replacement; *CPB*, cardiopulmonary bypass; *CABG*, coronary artery bypass grafting; *SD*, standard deviation.

Table 3. Outcomes

Variable	All ARE	All AVR	*P* value	Isolated ARE	Isolated AVR	*P* value
Stroke/TIA	8 (3.2%)	66 (3.1%)	.8485	6 (3.9%)	25 (2.4%)	.2754
Reoperation for bleeding	12 (4.8%)	96 (4.5%)	.8721	4 (4.3%)	39 (3.7%)	.7731
Perioperative myocardial infarction	1 (0.4%)	7 (0.3%)	.5898	0 (0.0%)	2 (0.2%)	1.000
Pacemaker	10 (3.6%)	76 (3.6%)	.7197	8 (5.1%)	36 (3.4%)	.2580
Operative mortality	14/249 (5.6%)	61/2117 (2.9%)	.0324	7/156 (4.5%)	23/1054 (2.2%)	.0953

ARE, Aortic root enlargement; *AVR*, aortic valve replacement; *TIA*, transient ischemic attack.

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each of the very large number of valvular prostheses in clinical use. In an effort to account for this, a sophisticated analysis performed by investigators at the Cleveland Clinic using multivariable propensity scores and multivariable hazard function analyses with bootstrap resampling defined PPM in no less than 4 different manners, including manufacturers' labeled valve size, manufacturers' stated internal orifice area, indexed internal orifice area, and disease score as an expression of variant of internal orifice area from the expected value.[12] Regardless, it is intuitive that an operation performed to relieve valvular stenosis should leave the patient with the least possible residual obstruction to flow. It is also clear that transvalvular gradients increase exponentially as the iEOA decreases to less than 0.8 to 0.9 cm^2/m^2.[22]

Table 4. Predictors of operative mortality

	Univariate		Multivariate	
Variable	*P* value	OR	*P* value	OR
Age	.0044	1.03		
Female sex	.0323	1.65		
ARE	.0219	2.01		
Redo	.4474	1.34		
BSA (per unit decrease)	.0500	2.59		
BMI	.5774	1.01		
Myocardial infarction	.0236	1.93		
Congestive heart failure	<.0001	4.62	<.0001	3.22
Previous operation	.7572	1.12		
Previous AV surgery	.8507	1.09		
NSR (= no)	.0096	2.26		
NYHA	<.0001	2.52	.0020	1.87
LVEF (per unit decrease)	<.0001	1.03		
Rheumatic	.8182	1.13		
Bicuspid AV (= no)	.0009	3.12		
Calcific AV (= no)	.7604	1.11		
Urgent/emergency status	.0619	2.15		
Valve implant size (per unit decrease)	.0005	1.22	.012	1.16
Creatinine >2.0 mg/dL	.4958	1.19		

OR, Odds ratio; *ARE*, aortic root enlargement; *BSA*, body surface area; *BMI*, body mass index; *AV*, aortic valve; *NSR*, normal sinus rhythm; *NYHA*, New York Heart Association; *LVEF*, left ventricular ejection fraction.

Choice among the 3 common techniques of root enlargement can be dictated by individual surgeon experience, as well as complexity inherent to the procedure. The Konno–Rastan procedure[23,24] offers the greatest degree of root enlargement. It is a complex procedure, however, requiring creation of a ventricular septal defect and right ventriculotomy, with double-patch closure of both. This risks damage to the septal arteries, as well as the conduction system, and places the patient at risk of intercameral fistulae. The posterior root enlargement techniques described by Nicks and colleagues[7] and Manouguian and Seybold-Epting[17] are more straightforward technically, although arguments have been made concerning impedance to outflow imposed by angular distortion of the left ventricular outflow tract with overriding of the prosthesis on the anterior leaflet of the mitral valve. Choice between Nicks' and Manouguian's enlargement will likely be largely dictated by the surgeon's preferred aortotomy, oblique or transverse, with the former enlargement representing an extension of the oblique and the latter an extension of the transverse approach (Figure 1). Both techniques as originally described cross the surgical annulus, although as commonly applied they might not.

Table 5A. Valve size ≤21 mm

Mortality	ARE	AVR	Total
No	143 (92.9%)	594 (95.5%)	737
Yes	11 (7.1%)	28 (4.5%)	39
Total	154	622	776

P = .2139. *ARE*, Aortic root enlargement; *AVR*, aortic valve replacement.

Table 5B. ARE ≤23 mm vs ARV ≤21 mm

Mortality	ARE, valve size <23 mm	AVR, valve size <21 mm	Total
No	218 (94%)	594 (95.5%)	812
Yes	14 (6%)	28 (4.5%)	42
Total	232	622	854

P = .3750. *ARE*, Aortic root enlargement; *AVR*, aortic valve replacement.

Our study had significant limitations. Despite being the largest study of its kind in the literature, the number of patients in the study remains relatively small. Our failure to assign statistical significance to the observed difference in mortality might therefore be due to insufficient power. Furthermore, because the study includes patients operated on by a large number of surgeons without a rigidly defined institutional philosophy regarding acceptable prosthesis size, details of the decision-making process regarding ARE are vague. This does not, however, weaken the empiric observations reported. Finally, as noted above, our data address only one half of the risk–benefit equation determining the indications for ARE. With absent data concerning the effect of PPM on hemodynamic outcome, we cannot argue the place of ARE in the surgeon's armamentarium nor justify its use in particular circumstances. Furthermore, we do not have hemodynamic measures of the effectiveness of posterior ARE in relieving the outflow gradient. Indeed, one could argue that the procedure as performed failed to provide sufficient annular enlargement because valve size remained a predictor of operative mortality. This might in part be due to inconsistency among surgeons with regard to extension of the enlargement across the true annulus. Nonetheless, our data do satisfactorily address the issue of incremental operative risk imposed by application of this approach.

We conclude that ARE using the Nicks technique can be accomplished with low operative risk, and accordingly, surgeons should not be reluctant to do so when they believe it is otherwise clinically indicated.

References

1. Gonzalez-Juanatey JR, Garcia-Acuna JM, Vega Fernandez M, Amaro Cendon A, Castelo Fuentes V, Garcia-Bengoechea JB, et al. Influence of the size of aortic valve prostheses on hemodynamics and change in left ventricular mass: implications for the surgical management of aortic stenosis. *J Thorac Cardiovasc Surg*. 1996;112:273-80.
2. Hirooka K, Kawazoe K, Kosakai Y, Sasako Y, Eishi K, Kito Y, et al. Prediction of postoperative exercise tolerance after aortic valve replacement. *Ann Thorac Surg*. 1994;58:1626-30.
3. Morris JJ, Schaff HV, Mullany CJ, Rastogi A, McGregor CG, Daly RC, et al. Determinants of survival and recovery of left ventricular function after aortic valve replacement. *Ann Thorac Surg*. 1993;56:22-30.
4. Rahimtoola SH. The problem of valve prosthesis–patient mismatch. *Circulation*. 1978;58:20-4.
5. Del Rizzo DF, Abdoh A, Cartier P, Doty D, Westaby S. Factors affecting left ventricular mass regression after aortic valve replacement with stentless valves. *Semin Thorac Cardiovasc Surg*. 1999;11:114-20.
6. Tasca G, Brunelli F, Cirillo M, DallaTomba M, Mhagna Z, Troise G, et al. Impact of valve prosthesis–patient mismatch on left ventricular mass regression following aortic valve replacement. *Ann Thorac Surg*. 2005;79:505-10.
7. Nicks R, Cartmill T, Bernstein L. Hypoplasia of the aortic root. The problem of aortic valve replacement. *Thorax*. 1970;25:339-46.
8. Blais C, Dumesnil JG, Baillot R, Simard S, Doyle D, Pibarot P. Impact of valve prosthesis–patient mismatch on short-term mortality after aortic valve replacement. *Circulation*. 2003;108:983-8.
9. Tasca G, Mhagna Z, Perotti S, Centurini PB, Sabatini T, Amaducci A, et al. Impact of prosthesis–patient mismatch on cardiac events and midterm mortality after aortic valve replacement in patients with pure aortic stenosis. *Circulation*. 2006;113:570-6.
10. Mohty-Echahidi D, Malouf JF, Girard SE, Schaff HV, Grill DE, Enriquez-Sarano ME, et al. Impact of prosthesis–patient mismatch on long-term survival in patients with small St Jude Medical mechanical prostheses in the aortic position. *Circulation*. 2006;113:420-6.
11. Koch CG, Khandwala F, Estafanous FG, Loop FD, Blackstone EH. Impact of prosthesis-patient size on functional recovery after aortic valve replacement. *Circulation*. 2005;111:3221-9.
12. Medalion B, Blackstone EH, Lytle BW, White J, Arnold JH, Cosgrove DM. Aortic valve replacement: is valve size important? *J Thorac Cardiovasc Surg*. 2000;119:963-74.
13. Hanayama N, Christakis GT, Mallidi HR, Joyner CD, Fremes SE, Morgan CD, et al. Patient prosthesis mismatch is rare after aortic valve replacement: valve size may be irrelevant. *Ann Thorac Surg*. 2002;73:1822-9.
14. Sommers KE, David TE. Aortic valve replacement with patch enlargement of the aortic annulus. *Ann Thorac Surg*. 1997;63:1608-12.
15. Stenseth JH, Danielson GK, McGoon DC. Pericardial patch enlargement of the aortic outflow tract. *J Thorac Cardiovasc Surg*. 1971;62:442-8.
16. Rankin JS, Hammill BG, Ferguson TB Jr, Glower DD, O'Brien SM, DeLong ER, et al. Determinants of operative mortality in valvular heart surgery. *J Thorac Cardiovasc Surg*. 2006;131:547-57.
17. Manouguian S, Seybold-Epting W. Patch enlargement of the aortic valve ring by extending the aortic incision into the anterior mitral leaflet. New operative technique. *J Thorac Cardiovasc Surg*. 1979;78:402-12.
18. Castro LJ, Arcidi JM Jr, Fisher AL, Gaudiani VA. Routine enlargement of the small aortic root: a preventive strategy to minimize mismatch. *Ann Thorac Surg*. 2002;74:31-6.
19. Kitamura M, Satoh M, Hachida M, Endo M, Hashimoto A, Koyanagi H. Aortic valve replacement in small aortic annulus with or without annular enlargement. *J Heart Valve Dis*. 1996;5(suppl 3):S289-93.
20. Rao V, Jamieson WR, Ivanov J, Armstrong S, David TE. Prosthesis–patient mismatch affects survival after aortic valve replacement. *Circulation*. 2000;102(suppl):III5-9.
21. Blackstone EH, Cosgrove DM, Jamieson WR, Birkmeyer NJ, Lemmer JH Jr, Miller DC, et al. Prosthesis size and long-term survival after aortic valve replacement. *J Thorac Cardiovasc Surg*. 2003;126:783-96.
22. Pibarot P, Dumesnil JG. Prosthesis–patient mismatch: definition, clinical impact, and prevention. *Heart*. 2006;92:1022-9.
23. Konno S, Imai Y, Iida Y, Nakajima M, Tatsuno K. A new method for prosthetic valve replacement in congenital aortic stenosis associated with hypoplasia of the aortic valve ring. *J Thorac Cardiovasc Surg*. 1975;70:909-17.
24. Rastan H, Koncz J. Aortoventriculoplasty: a new technique for the treatment of left ventricular outflow tract obstruction. *J Thorac Cardiovasc Surg*. 1976;71:920-7.

Discussion

Dr David A. Fullerton *(Denver, Colo)*. Dr Dhareshwar, I would like to compliment you both on a very crisp and clear presentation and also on an extremely well-written manuscript. Thank you very much for forwarding that to me, as well as a copy of your presentation.

I liked your topic, your presentation, and your article in large part because it fits my bias perfectly. I have to acknowledge that I have a very low threshold for enlarging the annulus, and therefore it is pleasing to see your data, particularly in light of the background literature on the subject.

Acknowledging that in your analysis the annular enlargement per se did not fall out as an independent risk factor for perioperative death, small valve size did. I am curious as to what your insight might be as to why that is the case.

Dr Dhareshwar. In our data set, if you look at the range of preoperative annulus sizes, almost 60 patients had an annulus size of less than 19 mm, which means we could not even get the 19-mm valve in place. Even after an ARE, the valve size that we could implant was 19 mm in almost 43 patients. With the large proportion of patients having a very small aortic annulus, in our study that turned out to be a more significant risk factor than annular enlargement per se.

Dr Fullerton. But according to your article, and I realize I am taking you beyond the analysis of this talk, among those who did not have an annular enlargement, there was a substantial percentage who actually received small valves. That led me to wonder what the indications were, if you know, for actually enlarging the annulus.

Dr Dhareshwar. I think it all depends on the clinical judgment at the time of the operation. Although everybody tries to implant the largest valve size possible, and we have in our minds when we go into the operating room a minimum size valve we are willing to place, sometimes it is not possible to enlarge the annulus. Presumably the surgeon believed annular enlargement was not needed in that particular patient.

Dr Fullerton. Given the fact that this is a retrospective review, am I safe to assume then that there is no protocol-driven algorithm for addressing the size of the anticipated implanted prosthesis as a function of the patient's body surface area?

Dr Dhareshwar. It is based on the individual surgeon. We always take into account the general body surface area and how the patient looks, his age, and the comorbidities. If the life expectancy is not that long—if we are doing a case on an 85-year-old patient, aortic annular enlargement might not be needed in that patient. It is a clinical judgment, and I think it has to be individualized to the patient.

Dr Fullerton. One final question. You indicated on your conclusion slide that you do not have long-term follow-up, but the literature would suggest that among patients who are given an AVR and who are left with an effective orifice area to body surface area ratio of less than about 0.8 cm^2/m^2, the classic definition of PPM, it turns out, to my surprise, that the survival curves for those 2 situations do not begin to diverge until about 7 or 8 years after the operation. Given the size of your database and the magnitude of your experience, I am curious to know whether you have any insight into that.

Dr Dhareshwar. We are working on that. It is one of the difficulties that we face when we look at the relationship between an effective orifice area rather than the valve size per se. Over the period of 9 years from 1993-2001, we had more than 15 different valves implanted. We have now restricted ourselves to 3 or 4 valve types, and it will be easier to predict the effective orifice area based on in vivo Doppler studies. We did not want to use geometric orifice areas, which we believe can be misleading. We would prefer to use iEOA based on Doppler studies in vivo. We hope that given the fewer valves that we have used over the last 8 to 10 years or so, in a couple of years we should have long-term data in these patients.

Dr Fullerton. Thank you very much.

Dr Dhareshwar. Thank you.

Dr D. Craig Miller *(Stanford, Calif)*. I have been trying to scratch the foggy catacombs of my brain, but you called this a Nicks, and if I remember correctly from 30 years ago, it is not a Nicks at all. A Nicks is done in the commissure between the right and the noncoronary sinus, the membranous septum. This is what most of us would call a hemi-Manouguian. Are there any historians or old-timers in the room who could clarify that?

Dr Sundt. Craig, there is often confusion on this. Manouguian's enlargement is through the commissure between the left and noncoronary sinus, and Nicks is at the base of the noncoronary sinus.

Dr Miller. He was an Australian surgeon, right? Okay. I stand corrected. Thank you. Manouguian does go through the commissure between the left and noncoronary sinus.

Dr Sundt. Does that go down in the record books at the Western? (laughter).

Dr Miller. I am wrong 100 times a day. So the Manouguian is between the commissure between the left and the noncoronary sinus, and the Nicks is right in the belly of the noncoronary sinus?

Dr Sundt. That is right.

Dr Miller. Okay, I stand corrected, and I think we all stand smarter.

Dr Sundt. Wait, the president says I am wrong, too. Scott?

Dr Mitchell. I hate to pull rank and disagree with Craig, but actually, the Manouguian I think stops just through the annulus, but the Nicks does not go onto the anterior leaflet. It stops before it goes across the annulus. Therefore this is probably a combination of the Nicks and Manouguian.

Unidentified speaker. I enjoyed your presentation. When you analyzed your data, did you look at patients who still had a residual mismatch after the enlargement, and did you analyze it with respect to mechanical versus stented tissue valves? Second, in the era of homografts and stentless tissue valves, what is your current indication for doing a root enlargement in these patients?

Dr Dhareshwar. That is an excellent question. Unfortunately, as I said before, we did not have a lot of data on the effective orifice areas, given more than 15 different valve types, and therefore we did not look at existing or residual PPM in these patients, but we hope that in subsequent studies we can look at that. There is no difference between the bioprosthesis and the mechanical valves.

Regarding homografts and stentless valves, our bias is toward performing root enlargement preferentially over stentless valves. We currently use homografts in the pediatric population and in adults only if we are faced with the situation of endocarditis. Very rarely do we use stentless valves when we think that the patient needs a root replacement for aneurysm on dissection rather than just an AVR, and therefore we are biased toward a root enlargement and getting in a larger valve than using stentless valves.

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Dr Benjamin Youdelman *(Philadelphia, Pa)*. In your study you looked at 15 different types of valves. The data I am presenting at this meeting comparing 8 common valves shows a range of −1.5 to +0.5 mm between the measured size and the labeled valve size, and I suspect there would be a similar range over your 15 different valves. I would argue that your analysis should not be done using labeled valve size but that you should use geometric orifice area. I would also argue that effective orifice area should not be used because of the variability in measurements that is likely caused by differences in heart rate, blood pressure, ventricular function, and echocardiographic data acquisition.

Dr Dhareshwar. I agree with your comments. I also had a look at your poster. It is a nice poster.